REMARKS

Claims 56-98 are pending in the instant application. Claims 67 and 70 have been withdrawn from consideration by the Examiner. Claims 56-66, 68-69 and 71-98 have been rejected. Claims 56, 57, 62, 63, 65, 67, 68, 69, 76, 77, 78, 80, 81, 82, 87, 88, 90, 97 and 98 have been amended. Claims 60, 61, 70, 85 and 86 have been canceled without prejudice. Support for these amendments is provided in the specification at page 10, line 23 through page 11, line 9 and in canceled claims 60-61 and 85-86. No new matter is added by these claims. Reconsideration is respectfully requested in light of these amendments and the following remarks.

I. Scope of Examination and Claim Amendments in Response Thereto

The Examiner suggests that claims 56-98 are directed to inventions that are independent and distinct from the invention originally claimed. In particular, the Examiner suggests that Applicant's election in March of 2000 was to claims to methods employing antibodies and functional fragments thereof. Further, the Examiner suggests that Applicant's election in March of 2000 was to claims to methods employing troponin I and residues 1-193 of troponin

I and myosin light chain 1 peptide fragment comprising residues 20-199. In addition, the Examiner suggests that Applicant's election in December of 2001 was to claims to methods of assessing muscle damage by assessing modification of peptide fragments of troponin I.

Accordingly, in an earnest effort to advance the prosecution of this case, Applicants have amended the claims to be drawn to methods employing antibodies and functional fragments thereof.

Further, it is respectfully pointed out that elections made by Applicant in December 2001 were species elections made with traverse. In accordance with 37 C.F.R. 1.141, more than one species of an invention, not to exceed a reasonable number, may be specifically claimed in different claims in one national application provided that the application includes an allowable claim generic to all the claimed species and all the claims to species in excess of one are written in dependent form. Applicants believe amendments to the claims set forth in the instant action place generic claims to methods of the present invention for a reasonable number of species in condition for allowance and thus further restriction to a single species is neither required nor proper. See MPEP 809.02 and 37 C.F.R. 1.141.

Entry of these amendments to the claims encompassing a reasonable number of species in accordance with MPEP 809.02 is therefore respectfully requested.

II. Objection under 35 U.S.C. 132

The amendment filed August 6, 2004 has been objected to under 35 U.S.C. 132 as introducing new matter.

In particular, the Examiner suggests that material added at paragraphs beginning at page 9, line 21, page 29, line 4 and page 32, line 19 relating to the epitope TnI amino acid residues are not supported by the original disclosure.

Further, the Examiner suggests that no explanation has been provided as to the change in residue numbering with respect to the MLC1 peptide fragment in paragraphs beginning at page 10, line 21; page 12, line 14; page 14, line 3; page 24, line 12 and page 25, line 4.

MPEP 2163.07 states that an amendment to correct an obvious error does not constitute new matter where one skilled in the art would not only recognize the existence of the error in the specification but also the appropriate correction. Also see In re Oda, 443 F.2d 1200, 170 USPQ 268 (CCPA 1971).

Accordingly, in an earnest effort to advance the prosecution of this case, Applicants are providing herewith a copy of the web page for anti-cardiac TnI MAb 8I-7 from Spectral Diagnostics disclosing the epitope for this antibody to be amino acid residues 137 to 148. It is taught at page 29, line 21 that the anti-TnI antibody was obtained from Spectral Diagnostics. Thus, the amendment to paragraphs beginning at page 9, line 21, page 29, line 4 and page 32, line 19 relating to the epitope TnI amino acid residues was made to correct an obvious error in the specification.

Further, Applicants have amended the specification at paragraphs beginning at page 10, line 21, page 12, line 14 and page 14, line 3, to replace "192" with --199--.

In addition, Applicants are providing an amended Sequence Listing herewith. Please replace the current Sequence Listing with the amended Sequence Listing provided herewith. Applicants are also providing herewith a Statement that the provided CRF copy of the Sequence Listing is identical to the amended paper copy of the Sequence Listing submitted herewith.

This sequence listing has been amended to be inclusive of both the MLC1 rat sequence of 199 amino acids as SEQ ID NO:28 and the corresponding shorter human MLC1 sequence

referred to by Zimmermann et al. (J. Mol. Biol. 1990 211(3):505-513) by reference to Kurabayashi et al. J. Biol. Chem. 1988 263:13930 as SEQ ID NO:50, both of which were in the public domain at the time of filing the instant application. As made clear at page 25, lines 9-11, contents of all references cited throughout the application are expressly incorporated by reference. Applicants have amended the specification to include the correct reference citations for the publicly available rat and human MLC1 sequences and are providing copies of these references herewith.

Each of these amendments was made to correct an obvious error in the specification noted by a skilled artisan upon his review of the specification in preparation of this response and the response filed August 6, 2004 and constitute the correction the skilled artisan considered appropriate for this obvious error.

Thus, in accordance with MPEP 2163.07 these amendments do not constitute new matter and their entry as well as withdrawal of all pending objections is respectfully requested.

III. Rejection of Claim 69 under 35 U.S.C. 112, first paragraph - Lack of Written Description

Claim 69 has been rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. In particular, the Examiner suggests that SEQ ID NO:28 is a new amino acid sequence.

As discussed in Section II supra, the specification and Sequence Listing have been amended to address the new matter rejection.

Further, claim 69 has been amended and no longer contains reference to SEQ ID NO:28.

Withdrawal of this rejection is therefore respectfully requested.

IV. Rejection of Claims 56-59, 71-84, 94 and 96-98 under 35 U.S.C. 112, second paragraph

Claims 56-59, 71-84, 94 and 96-98 have been rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. In particular, the Examiner suggests that without a recitation of the specific process steps involved in detecting, the metes and bounds of the claims are indefinite

because it cannot be determined what is specifically encompassed by the method of detection.

Applicants respectfully disagree.

MPEP § 2173.02 states that definiteness of claim language must be analyzed not in a vacuum, but in light of: (A) the content of the particular application; (B) the teachings of the prior art; and (C) the claim interpretation that would be given by one possessing the ordinary level of skill in the pertinent art at the time the invention was made. Steps involved in detecting the presence or absence or measuring the amount of a peptide fragment of a myofilament protein or a covalent or non-covalent complex of at least a peptide fragment of a myofilament protein and an intact myofilament protein or two peptide fragments of myofilament proteins in a biological sample obtained from a subject would be clear to one of skill in the art when read in light of the teachings of the specification, for example at page 13, line 27 and extending to page 14, line 19. Thus, further clarification of such methods steps in the claims should not be required given the content of the application.

However, in an earnest effort to advance the prosecution, Applicants have amended claims 56, 80 and 97 to

include method steps for detection as set forth in dependent claims 60-61 and 85-86, now canceled.

Withdrawal of these rejections is therefore is respectfully requested.

V. Rejection of Claims 56-65, 68-69, 71-90 and 92-98 under 35 U.S.C. 102(b)

Claims 56-65, 68-69, 71-90 and 92-98 have been rejected under 35 U.S.C. § 102(b) as being anticipated by Lofberg et al. The Examiner suggests that Lofberg discloses use of various antibodies and detectable labels and markers to detect two different fragments of myosin heavy-chain, troponin I and troponin T for the purpose of assaying acute muscle damage, irreversible cardiac and skeletal muscle damage and reversible skeletal muscle damage from biological samples such as serum. The Examiner suggests that "when an antibody binds to two different myosin heavy chain fragments and troponin proteins as it does in the Lofberg reference, it meets the claim limitation of "a peptide fragment of a myofilament protein and in intact protein"."

Claims 56-65, 68-69, 71-90 and 92-98 have also been rejected under 35 U.S.C. 102(b) as being anticipated by Westfall et al. The Examiner suggests that Westfall discloses use of various antibodies and detectable markers

to detect fragments from both troponin I and troponin T for the purpose of assaying cardiac muscle damage from ischemia from biological samples such as a component of cardiac muscle tissue. Further, the Examiner suggests that "when an antibody binds to two different troponin fragments (such as from troponin I and troponin T) as it does in the Westfall reference, it meets the limitations of "a peptide fragment of a myofilament proteins and an intact protein" because the intact protein is the antibody and a peptide fragment includes "all or a portion of a cardiac troponin I fragment"."

Claims 56-66, 68-69 and 71-98 have also been rejected under 35 U.S.C. 102(b) as being anticipated by Wicks et al. (WO 94/27156). The Examiner suggests that Wicks discloses the use of antibodies and detectable labels and markers to detect troponin I (and specific fragments, page 5 and claims 12-13, 18, 26-27, 32-34 and 36) and troponin C in a complex in sandwich assays having immobilized solid phases for the purpose of assaying irreversible cardiac damage from biological samples such as blood. Further, the Examiner suggests that "when an antibody binds to two different troponin fragments (such as troponin I and troponin C) as it does in the Wicks reference, it meets the limitations of "a peptide fragment of a myofilament protein and an intact

protein" because the intact protein is the antibody and a peptide fragment of a myofilament protein includes "all or a portion of a cardiac troponin I peptide fragment"."

Claims 56, 60-66, 68-69 and 71-79 have been rejected under 35 U.S.C. 102(b) as being anticipated by Takahashi et al. (WO 96/10078). The Examiner suggests that Takahashi discloses the use of antibodies and detectable labels and markers to detect myosin light chain 1 (MLC-1) in a complex in sandwich assays having immobilized solid phases (pages 10 and 12) for the purpose of assaying cardiac damage from biological samples such as blood (pages 2-5). Further, the Examiner suggests that "when an antibody binds to MLC-1 as it does in the Takahashi reference, it meets the limitations of a "peptide fragment of a myofilament protein and an intact protein" because the intact protein is the antibody and a peptide fragment of a myofilament protein includes "all or a portion of a cardiac troponin I fragment"."

Accordingly, in an earnest effort to advance the prosecution of this case, Applicants have amended the claims in accordance with teachings at page 10, line 23 through page 11, line 9, line to state that the intact protein is an intact myofilament protein. No new matter is added by this amendment.

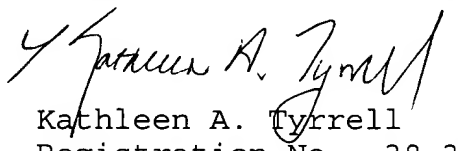
This amendment clearly distinguishes the present invention from teachings of Lofberg, Westfall, Wicks and Takahashi wherein the intact protein is not an intact myofilament protein, but rather, as stated by the Examiner, an antibody.

Withdrawal of these rejections under 35 U.S.C. § 102(b) is therefore respectfully requested.

VI. Conclusion

Applicants believe that the foregoing comprises a full and complete response to the Office Action of record. Accordingly, favorable reconsideration and subsequent allowance of the pending claims is earnestly solicited.

Respectfully submitted,



Kathleen A. Tyrrell
Registration No. 38,350

Date: **March 28, 2005**

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UniProtKB/Swiss-Prot entry P16409

Entry information

Entry name	**MYL3_RAT**
Primary accession number	**P16409**
Secondary accession numbers	None
Entered in Swiss-Prot in	Release 15, August 1990
Sequence was last modified in	Release 15, August 1990
Annotations were last modified in	Release 47, May 2005

Name and origin of the protein

Protein name	**Myosin light polypeptide 3**
Synonyms	**Myosin light chain 1, slow-twitch muscle B/ventricular isoform** **MLC1SB** **Ventricular/slow twitch myosin alkali light chain**
Gene name	**Name: Myl3** Synonyms: Mlc1v
From	Rattus norvegicus (Rat) [TaxID: 10116]
Taxonomy	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia; Sciurognathi; Muridae; Murinae; Rattus.

References

[1] NUCLEOTIDE SEQUENCE.
TISSUE=Heart ventricle;
MEDLINE=89240031;PubMed=2717409
McNally E., Buttrick P., Leinwand L.;
"Ventricular myosin light chain 1 is developmentally regulated and does not change in hypertension.";
Nucleic Acids Res. 17:2753-2767(1989).

[2] NUCLEOTIDE SEQUENCE.
STRAIN=Wistar;
TISSUE=Heart ventricle;
MEDLINE=90016857;PubMed=2798124
Periasamy M., Wodgaonkar R., Kumar C., Martin B.J., Siddiqui M.A.Q.;
"Characterization of a rat myosin alkali light chain gene expressed in ventricular and slow twitch skeletal muscles.";
Nucleic Acids Res. 17:7723-7734(1989).

[3] NUCLEOTIDE SEQUENCE [LARGE SCALE MRNA].
TISSUE=Heart;
NIH - Mammalian Gene Collection (MGC) project;
Submitted (SEP-2004) to the EMBL/GenBank/DDBJ databases.

Comments

- ***FUNCTION***: Regulatory light chain of myosin. Does not bind calcium.
- ***SUBUNIT***: Myosin is an hexamer of 2 heavy chains and 4 light chains.

- ***SIMILARITY***: Contains 2 EF-hand calcium-binding domains.

Copyright

Cross-references

EMBL	X14812; CAA32917.1; -. X16325; CAA34388.1; -. X16326; CAA34388.1; JOINED. X16327; CAA34388.1; JOINED. X16329; CAA34388.1; JOINED. X16330; CAA34388.1; JOINED. BC081832; AAH81832.1; -.
PIR	S09573; MORT3V.
HSSP	P02607; 1BR1.
Rat-heart-2DPAGE	P16409; -.
RGD	3142; Myl3.
GeneLynx	Myl3; Rattus norvegicus.
InterPro	IPR002048; EF-hand. IPR010983; EF_Hand_like.
Pfam	PF00036; efhand; 1.
ProDom	PD000012; EF-hand; 2.
SMART	SM00054; EFh; 2.
Implicit links to	HOVERGEN; BLOCKS; ProtoNet; ProtoMap; PRESAGE; DIP; ModBase; SMR; SWISS-2DPAGE; UniRef.

Keywords

Motor protein; **Multigene family**; **Muscle protein**; **Myosin**; **Repeat**.

Features

Key	From	To	Length	Description
INIT_MET	0	0		
DOMAIN	66	79	14	Ancestral calcium site 1.
DOMAIN	145	156	12	Ancestral calcium site 2.

Sequence information

Length: **199 AA**
Molecular weight: **22025 Da**
CRC64: **CCA4758A7A3F1D53** [This is a checksum on the sequence]

```
        10         20         30         40         50         60
APKKPEPKKD DAKTAAPKAA PAPAAAPAAA PEPERPKEAE FDASKIKIEF TPEQIEEFKE


h       e       g cg  b    ce          b e  c
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        70         80         90        100        110        120
AFQLFDRTPK GEMKITYGQC GDVLRALGQN PTQAEVLRVL GKPKQEELNS KMMDFETFLP

       130        140        150        160        170        180
MLQHISKNKD TGTYEDFVEG LRVFDKEGNG TVMGAELRHV LATLGERLTE DEVEKLMAGQ

       190
EDSNGCINYE AFVKHIMAS
```

h e g cg b ce b e c

UniProtKB/Swiss-Prot entry P08590

Entry information

Entry name	**MYL3_HUMAN**
Primary accession number	**P08590**
Secondary accession number	Q9NRS8
Entered in Swiss-Prot in	Release 08, August 1988
Sequence was last modified in	Release 21, March 1992
Annotations were last modified in	Release 47, May 2005

Name and origin of the protein

Protein name	**Myosin light polypeptide 3**
Synonyms	**Myosin light chain 1, slow-twitch muscle B/ventricular isoform** **MLC1SB** **Ventricular/slow twitch myosin alkali light chain** **Cardiac myosin light chain-1** **CMLC1**
Gene name	**Name: MYL3**
From	Homo sapiens (Human) [TaxID: 9606]
Taxonomy	Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Primates; Catarrhini; Hominidae; Homo.

References

[1] NUCLEOTIDE SEQUENCE.
MEDLINE=88189843;PubMed=3357795
Hoffmann E., Shi Q.W., Floroff M., Mickle D.A.G., Wu T.-W., Olley P.M., Jackowski G.;
"Molecular cloning and complete nucleotide sequence of a human ventricular myosin light chain 1.";
Nucleic Acids Res. 16:2353-2353(1988).

[2] SEQUENCE REVISION.
Jackowski G.;
Submitted (MAY-1988) to the EMBL/GenBank/DDBJ databases.

[3] NUCLEOTIDE SEQUENCE.
MEDLINE=88330932;PubMed=3417683
Kurabayashi M., Komuro I., Tsuchimochi H., Takaku F., Yazaki Y.;
"Molecular cloning and characterization of human atrial and ventricular myosin alkali light chain cDNA clones
J. Biol. Chem. 263:13930-13936(1988).

[4] NUCLEOTIDE SEQUENCE.
MEDLINE=89123281;PubMed=2789520
Fodor W.L., Darras B., Seharaseyon J., Falkenthal S., Francke U., Vanin E.F.;
"Human ventricular/slow twitch myosin alkali light chain gene characterization, sequence, and chromosomal location.";
J. Biol. Chem. 264:2143-2149(1989).

[5] NUCLEOTIDE SEQUENCE.
Huang R., Peng B., Zhou G., Gong Z.;
"The sequence of human cardiac myosin light chain I (CMLC-1) from a Chinese patient and the preparation of

h e g cg b ce b e c

monoclonal antibody to CHCMLC1.";
Submitted (AUG-1999) to the EMBL/GenBank/DDBJ databases.

[6] NUCLEOTIDE SEQUENCE [LARGE SCALE MRNA].
TISSUE=Skeletal muscle;
DOI=10.1073/pnas.242603899;MEDLINE=22388257;PubMed=12477932
Strausberg R.L., Feingold E.A., Grouse L.H., Derge J.G., Klausner R.D., Collins F.S., Wagner L., Shenmen C.M Schuler G.D., Altschul S.F., Zeeberg B., Buetow K.H., Schaefer C.F., Bhat N.K., Hopkins R.F., Jordan H., Moo T., Max S.I., Wang J., Hsieh F., Diatchenko L., Marusina K., Farmer A.A., Rubin G.M., Hong L., Stapleto M., Soares M.B., Bonaldo M.F., Casavant T.L., Scheetz T.E., Brownstein M.J., Usdin T.B., Toshiyuki S., Carninci P., Prange C., Raha S.S., Loquellano N.A., Peters G.J., Abramson R.D., Mullahy S.J., Bosak S.A., McEwan P.J., McKernan K.J., Malek J.A., Gunaratne P.H., Richards S., Worley K.C., Hale S., Garcia A.M., Ga L.J., Hulyk S.W., Villalon D.K., Muzny D.M., Sodergren E.J., Lu X., Gibbs R.A., Fahey J., Helton E., Kettema M., Madan A., Rodrigues S., Sanchez A., Whiting M., Madan A., Young A.C., Shevchenko Y., Bouffard G.G., Blakesley R.W., Touchman J.W., Green E.D., Dickson M.C., Rodriguez A.C., Grimwood J., Schmutz J., Myers R.M., Butterfield Y.S.N., Krzywinski M.I., Skalska U., Smailus D.E., Schnerch A., Schein J.E., Jones S.J.M. , Marra M.A.;
"Generation and initial analysis of more than 15,000 full-length human and mouse cDNA sequences.";
Proc. Natl. Acad. Sci. U.S.A. 99:16899-16903(2002).

[7] PROTEIN SEQUENCE OF 19-26; 33-39; 94-99; 123-128; 138-141 AND 154-170.
TISSUE=Heart;
MEDLINE=96007936;PubMed=7498159
Kovalyov L.I., Shishkin S.S., Efimochkin A.S., Kovalyova M.A., Ershova E.S., Egorov T.A., Musalyamov A.K
"The major protein expression profile and two-dimensional protein database of human heart.";
Electrophoresis 16:1160-1169(1995).

[8] VARIANTS MVC1 VAL-148 AND HIS-153.
MEDLINE=96241574;PubMed=8673105
Poetter K., Jiang H., Hassanzadeh S., Master S.R., Chang A., Dalakas M.C., Rayment I., Sellers J.R., Fananapa L., Epstein N.D.;
"Mutations in either the essential or regulatory light chains of myosin are associated with a rare myopathy in human heart and skeletal muscle.";
Nat. Genet. 13:63-69(1996).

[9] VARIANT CMH8 LYS-142.
PubMed=12021217
Olson T.M., Karst M.L., Whitby F.G., Driscoll D.J.;
"Myosin light chain mutation causes autosomal recessive cardiomyopathy with mid-cavitary hypertrophy and restrictive physiology.";
Circulation 105:2337-2340(2002).

[10] VARIANT CMH8 GLY-55.
DOI=10.1161/01.CIR.0000066323.15244.54;PubMed=12707239
Richard P., Charron P., Carrier L., Ledeuil C., Cheav T., Pichereau C., Benaiche A., Isnard R., Dubourg O., Burban M., Gueffet J.-P., Millaire A., Desnos M., Schwartz K., Hainque B., Komajda M.;
"Hypertrophic cardiomyopathy: distribution of disease genes, spectrum of mutations, and implications for a molecular diagnosis strategy.";
Circulation 107:2227-2232(2003).

[11] ERRATUM.
Richard P., Charron P., Carrier L., Ledeuil C., Cheav T., Pichereau C., Benaiche A., Isnard R., Dubourg O., Burban M., Gueffet J.-P., Millaire A., Desnos M., Schwartz K., Hainque B., Komajda M.;
Circulation 109:3258-3258(2004).

Comments

- ***FUNCTION***: Regulatory light chain of myosin. Does not bind calcium.
- ***SUBUNIT***: Myosin is an hexamer of 2 heavy chains and 4 light chains.
- ***PTM***: The N-terminus is blocked.
- ***DISEASE***: Defects in MYL3 are the cause of familial hypertrophic cardiomyopathy 8 (CMH8) [MIM:608751 192600]; also designated HCM or FHC. Hypertrophic cardiomyopathy is a heart disorder characterized by ventricular hypertrophy, which is usually asymmetric and often involves the interventricular septum. The prevalence of the disease in the general population is 0.2%. FHC is clinically heterogeneous, with inter- and intrafamilial variations ranging from benign to maligmant forms with high risk of cardiac failure and sudden cardiac death. CMH8 inheritance can be autosomal dominant or recessive.
- ***DISEASE***: Defects in MYL3 are the cause of hypertrophic cardiomyopathy with mid-left ventricular chamber type 1 (MVC1) [MIM:608751]. MVC1 is a very rare variant of familial hypertrophic cardiomyopathy, characterized by mid-left ventricular chamber thickening.
- ***SIMILARITY***: Contains 2 EF-hand calcium-binding domains.
- ***DATABASE***: NAME=Familial hypertrophic cardiomyopathy mutation database; WWW="http://www.angis.org.au/Databases/Heart/heartbreak.html".

Copyright

Cross-references

EMBL	M24247; AAA59851.1; -. M24242; AAA59851.1; JOINED. M24243; AAA59851.1; JOINED. M24244; AAA59851.1; JOINED. M24245; AAA59851.1; JOINED. M24246; AAA59851.1; JOINED. X07373; CAA30292.1; -. M24122; AAA59895.1; -. AF174483; AAF91089.1; -. BC009790; AAH09790.1; -.
PIR	B30881; MOHU3V.
HSSP	P02607; 1BR1.
HSC-2DPAGE	P08590; HUMAN.
Genew	HGNC:7584; MYL3.
CleanEx	HGNC:7584; MYL3.
GeneCards	MYL3.
GeneLynx	MYL3; Homo sapiens.
GenAtlas	MYL3.
H-InvDB	HIX0003256; -.
MIM	160790 . 608751 . 192600 .

GO	GO:0005859;Cellular component: muscle myosin *(traceable author statement)*. GO:0008307;Molecular function: structural constituent of muscle *(traceable author statement)*.
InterPro	IPR002048; EF-hand. IPR010983; EF_Hand_like.
Pfam	PF00036; efhand; 1.
ProDom	PD000012; EF-hand; 2.
Implicit links to	SOURCE; HOVERGEN; BLOCKS; ProtoNet; ProtoMap; PRESAGE; DIP; ModBase; SMR; SWIS 2DPAGE; UniRef.

Keywords

Cardiomyopathy; Direct protein sequencing; Disease mutation; Motor protein; Multigene family; Muscle protein; Myosin; Repeat.

Features

Key	From	To	Length	Description	FTId
INIT_MET	0	0		By similarity.	
DOMAIN	61	74	14	Ancestral calcium site 1.	
DOMAIN	140	151	12	Ancestral calcium site 2.	
VARIANT	55	55	1	E -> G (in CMH8).	VAR_019842
VARIANT	142	142	1	E -> K (in CMH8; autosomal recessive).	VAR_019843
VARIANT	148	148	1	M -> V (in MVC1).	VAR_004599
VARIANT	153	153	1	R -> H (in MVC1).	VAR_004600
CONFLICT	170	170		K -> R (in Ref. 5).	

Sequence information

Length: **194 AA**
Molecular weight: **21801 Da**
CRC64: **A933F3874D812972** [This is a checksum on the sequence]

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        70         80         90        100        110        120
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       130        140        150        160        170        180
SKNKDTGTYE DFVEGLRVFD KEGNGTVMGA ELRHVLATLG ERLTEDEVEK LMAGQEDSNG

       190
CINYEAFVKH IMSS
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h e g cg b ce b e c